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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/692,439	10/22/2003	David J. Pinsky	51917-CB-PCT-US/JPW/AJM/A	8415

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EXAMINER	
SZPERKA, MICHAEL EDWARD	
ART UNIT	PAPER NUMBER
1644	

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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/692,439	PINSKY ET AL.	
	Examiner	Art Unit	
	Michael Szperka	1644	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *26 January 2009*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *25* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *25* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *10/22/08*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

1. Applicant's arguments and claim amendments received January 26, 2009 are acknowledged.

Claims 1-24 and 26-35 have been canceled.

Claim 25 has been amended.

Claim 25 is under examination as it reads on methods of treating reperfusion injuries by administering mutant factor IX molecules.

Information Disclosure Statement

2. The IDS received October 22, 2008 is acknowledged and has been considered.

Claim Rejections - 35 USC § 103

3. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

> This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

4. Claim 25 is rejected under 35 U.S.C. 103(a) as being unpatentable over Toledo-Pereyra (Klin Wochenschr, 1991, 69:1099-1104, of record) in view of Benedict et al. (of

record on the 9/20/04 IDS) in view of Rose et al. (WO 97/42900) and in view of Ludwig et al. (Blood, 1992, 79:1225-1232).

Toledo-Pereyra discloses that at the time the instant invention was made, a skilled artisan knew that "reperfusion injury" was often referred to by the descriptive physiological pathological process of thrombosis (see entire document, particularly the right column of page 1099). Toledo-Pereyra further discloses that fibrinogen activation and clotting (i.e. thrombosis) is a pathophysiological event in reperfusion injury that needs to be treated with pharmacological agents such as heparin to inhibit coagulation (see particularly the right column of page 1099, Table 7, and the right column of page 1103). These teachings differ from the instant claimed invention in that Toledo-Pereyra does not disclose the administration of "Factor IXa compounds" to treat thrombosis in reperfusion injury.

Benedict et al. disclose that inactivated Factor IXa was successfully used to inhibit thrombus formation in vivo (see entire document, particularly the abstract). They further disclose that administration of inactivated factor IXa offers an advantage over the administration of heparin for inhibiting coagulation in that animals treated with heparin suffered from excessive bleeding while animals given inactivated Factor IXa did not manifest excessive bleeding (see particularly figure 4). Benedict et al. disclose making inhibited factor IXa by incubating factor IXa with glu-gly-arg-chloromethyl ketone (see particularly the right column of page 1760).

Rose et al. disclose methods of inhibiting thrombus formation in vivo using inactivated factor IXa compounds, such compounds being disclosed as including factor IXa inactivated with glu-gly-arg-chloromethyl ketone as well as recombinant factor IX polypeptides wherein amino acids at the active site, particularly the active site serine, are mutated (see entire document, particularly pages 8 and 9).

Ludwig et al. disclose that the active site of factor IX comprises His221, Asp269 and Ser365, and further discloses multiple mutant factor IX polypeptides that are enzymatically inactive due to substitution mutations at these positions (see entire document, particularly the abstract).

Therefore, it would have been obvious to a person of ordinary skill in the art at the time the invention was made to administer inactivated Factor IXa to patients to treat reperfusion injury. Motivation to do so at the time the invention was made comes from the teachings of Toledo-Pereyra that thrombus formation in reperfusion injury is to be treated with heparin and the teachings of Benedict et al. that inactivated Factor IXa is better than heparin at inhibiting thrombus formation in vivo because unlike heparin, inactivated factor IXa administration does not lead to excessive bleeding. A person of ordinary skill in the art would have been further motivated to substitute factor IX polypeptides comprising active site substitution mutations, such as those disclosed by Rose et al. and Ludwig et al. for the chemically inactivated factor IX of Benedict et al. based upon the disclosure of Rose et al. that both chemically inactivated and recombinantly produced mutant factor IX polypeptides are to be used in methods of inhibiting coagulation. Note that this is because both the chemically inactivated and mutant factor IX polypeptides are enzymatically inactive. Additionally, Rose et al. explicitly state that "The factor IXa compound may be a recombinant Factor IXa compound in which amino acid**s** (emphasis by examiner) at the active site, especially at the active serine amino acid site, have been altered to render the recombinant factor IXa functionally inactive, but still capable of competing with intact, native factor IXa for cell surface binding." Given that the active site catalytic triad (Ser365, Asp269, His221) of factor IX was known in the art (see for example the last sentence of the first paragraph in the left column of page 1225 of Ludwig et al.) it is clear that Rose et al. contemplated the use of factor IX in which multiple active site members were simultaneously mutated.

Applicant's arguments filed January 26, 2009 have been fully considered but they are not persuasive. Applicant argues first that "reperfusion injury" is not synonymous with "thrombosis" and that there is no suggestion in the Toledo-Pereyra et al. reference that treating thrombosis is equivalent to treating reperfusion injury.

This argument is not persuasive. As applicant has pointed out, the art of Toledo-Pereyra et al. discloses that phenomenon of "reperfusion injury" has been referred to by many different descriptive pathological processes, one of which is thrombosis. Thus, Toledo-Pereyra et al. disclose that thrombosis is part of the complex pathological process presently identified as reperfusion injury, and that reperfusion injury has been treated by the administration of heparin (see the right column of page 1099 and Table 7 on page 1103). Thus, thrombosis is an intrinsic process which occurs during reperfusion injury. Given that Toledo-Pereyra et al. disclose the treatment of reperfusion injury with heparin and that thrombosis occurs during reperfusion injury, the disclosure of Benedict that enzymatically inactive factor IX is more effective than heparin at treating thrombosis, and the disclosure of Rose et al. that chemically inactivated and active site mutants of factor IX are all useful for treating thrombosis because they lack enzymatic activity, applicant's argument that the combination of references fails to teach treating reperfusion injury with factor IX mutants is not persuasive.

Applicant further argues that the specific combination of active site mutations recited in the instant claims is not disclosed in any of the cited references.

This argument is not persuasive because as stated in the rejection, Rose et al. disclose mutant factor IX molecules comprising mutations to the active site. The catalytic triad of factor IX was well known in the art at the time the instant invention and the disclosure of Rose et al. were made as evidenced by Ludwig et al., and as such it would have been obvious to a person of ordinary skill what additional mutations to the active site, in addition to the serine, could be mutated to make compounds comprising altered amino acid**s** at the active site which result in the enzymatic inactivity of the resulting compound.

5. Claim 25 is rejected under 35 U.S.C. 103(a) as being unpatentable over Toledo-Pereyra (Klin Wochenschr, 1991, 69:1099-1104, of record) in view of Benedict et al. (of

record on the 9/20/04 IDS) in view of US Patent 5,839,443 and in view of Ludwig et al. (Blood, 1992, 79:1225-1232).

Toledo-Pereyra discloses that at the time the instant invention was made, a skilled artisan knew that "reperfusion injury" was often referred to by the descriptive physiological pathological process of thrombosis (see entire document, particularly the right column of page 1099). Toledo-Pereyra further discloses that fibrinogen activation and clotting (i.e. thrombosis) is a pathophysiological event in reperfusion injury that needs to be treated with pharmacological agents such as heparin to inhibit coagulation (see particularly the right column of page 1099, Table 7, and the right column of page 1103). These teachings differ from the instant claimed invention in that Toledo-Pereyra does not disclose the administration of "Factor IXa compounds" to treat thrombosis in reperfusion injury.

Benedict et al. disclose that inactivated Factor IXa was successfully used to inhibit thrombus formation in vivo (see entire document, particularly the abstract). They further disclose that administration of inactivated factor IXa offers an advantage over the administration of heparin for inhibiting coagulation in that animals treated with heparin suffered from excessive bleeding while animals given inactivated Factor IXa did not manifest excessive bleeding (see particularly figure 4). Benedict et al. disclose making inhibited factor IXa by incubating factor IXa with glu-gly-arg-chloromethyl ketone (see particularly the right column of page 1760).

The '443 patent discloses methods of inhibiting thrombus formation in vivo using inactivated factor IXa compounds, such compounds being disclosed as including factor IXa inactivated with glu-gly-arg-chloromethyl ketone as well as recombinant factor IX polypeptides wherein amino acids at the active site, particularly the active site serine, are mutated (see entire document, particularly column 4).

Ludwig et al. disclose that the active site of factor IX comprises His221, Asp269 and Ser365, and further discloses multiple mutant factor IX polypeptides that are enzymatically inactive due to substitution mutations at these positions (see entire document, particularly the abstract).

Therefore, it would have been obvious to a person of ordinary skill in the art at the time the invention was made to administer inactivated Factor IXa to patients to treat reperfusion injury. Motivation to do so at the time the invention was made comes from the teachings of Toledo-Pereyra that thrombus formation in reperfusion injury is to be treated with heparin and the teachings of Benedict et al. that inactivated Factor IXa is better than heparin at inhibiting thrombus formation in vivo because unlike heparin, inactivated factor IXa administration does not lead to excessive bleeding. A person of ordinary skill in the art would have been further motivated to substitute factor IX polypeptides comprising active site substitution mutations, such as those disclosed by Ludwig et al. for the chemically inactivated factor IX of Benedict et al. based upon the disclosure of the '443 patent that both chemically inactivated and recombinantly produced mutant factor IX polypeptides are to be used in methods of inhibiting coagulation. Note that this is because both the chemically inactivated and mutant factor IX polypeptides are enzymatically inactive. Additionally, the '443 patent explicitly states that "The factor IXa compound may be a recombinant Factor IXa compound in which amino acid**s** (emphasis by examiner) at the active site, especially at the active serine amino acid site, have been altered to render the recombinant factor IXa functionally inactive, but still capable of competing with intact, native factor IXa for cell surface binding." Given that the active site catalytic triad (Ser365, Asp269, His221) of factor IX was known in the art (see for example the last sentence of the first paragraph in the left column of page 1225 of Ludwig et al.) it is clear that the '443 patent contemplates the use of factor IX in which multiple active site members were simultaneously mutated.

Applicant's arguments filed January 26, 2009 have been fully considered but they are not persuasive. Applicant's arguments are the same as above since Rose et al. and the '443 patent are the same disclosure, differing from one another only in their pagination and date of availability as prior art. These arguments were not found persuasive for the reasons discussed supra, and thus the rejection is maintained.

Double Patenting

6. The nonstatutory double patenting rejection is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the "right to exclude" granted by a patent and to prevent possible harassment by multiple assignees. A nonstatutory obviousness-type double patenting rejection is appropriate where the conflicting claims are not identical, but at least one examined application claim is not patentably distinct from the reference claim(s) because the examined application claim is either anticipated by, or would have been obvious over, the reference claim(s). See, e.g., *In re Berg*, 140 F.3d 1428, 46 USPQ2d 1226 (Fed. Cir. 1998); *In re Goodman*, 11 F.3d 1046, 29 USPQ2d 2010 (Fed. Cir. 1993); *In re Longi*, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); *In re Van Ornum*, 686 F.2d 937, 214 USPQ 761 (CCPA 1982); *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970); and In *re Thorington*, 418 F.2d 528, 163 USPQ 644 (CCPA 1969).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321(c) or 1.321(d) may be used to overcome an actual or provisional rejection based on a nonstatutory double patenting ground provided the conflicting application or patent either is shown to be commonly owned with this application, or claims an invention made as a result of activities undertaken within the scope of a joint research agreement.

Effective January 1, 1994, a registered attorney or agent of record may sign a terminal disclaimer. A terminal disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b).

7. Claim 25 is rejected on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claims 1-19 of U.S. Patent No. 6,316,403 in view of Toledo-Pereyra (of record) in view of Benedict et al. (of record) in view of Rose et al. (WO 97/42900) and in view of Ludwig et al. (Blood, 1992, 79:1225-1232).

The claims of the '403 patent recite methods of treating ischemic disorders by administering inactivated factor IX to a patient to inhibit coagulation so as to treat the ischemic disorder in the patient. The claims of the '403 patent differ from the instant claimed invention in that the claims of the '403 patent do not specifically recite reperfusion injury and do not recite that the species of inactivated Factor IX recited in the instant claim.

Toledo-Pereyra discloses that at the time of the instant invention, a skilled artisan would know that "reperfusion injury" is often referred to by the descriptive physiological pathological process of thrombosis (see entire document, particularly the right column of

page 1099). Toledo-Pereyra further discloses that fibrinogen activation and clotting (i.e. thrombosis) is a pathophysiological event in reperfusion (see particularly the right column of page 1099 and Table 7 on page 1103).

Benedict et al. disclose that factor IXa inactivated with glu-gly-arg-chloromethyl ketone successfully inhibits thrombus formation in vivo (see entire document, particularly the abstract).

Rose et al. disclose methods of inhibiting thrombus formation in vivo using inactivated factor IXa compounds, such compounds being disclosed as including factor IXa inactivated with glu-gly-arg-chloromethyl ketone as well as recombinant factor IX polypeptides wherein amino acids at the active site, particularly the active site serine, are mutated (see entire document, particularly pages 8 and 9).

Ludwig et al. disclose that the active site of factor IX comprises His221, Asp269 and Ser365, and further discloses multiple mutant factor IX polypeptides that are enzymatically inactive due to substitution mutations at these positions (see entire document, particularly the abstract).

Therefore, it would have been obvious to a person of ordinary skill in the art at the time the invention was made to administer factor IXa compounds, such as inactivated factor IX and IXa to treat reperfusion injuries. Motivation to do so comes from the teachings of the '403 patent which teaches that "Factor IXa" compounds are to be administered to inhibit thrombosis in a patient, and the teachings of Toledo-Pereyra that thrombosis is an important pathophysiological even that occurs in reperfusion injury. A person of skill in the art would be motivated to use a mutant factor IXa comprising active site mutations since Benedict et al. successfully inhibited thrombus formation in vivo using factor IX chemically inactivated using Glu-Gly-Arg chloromethyl ketone, since Rose et al. disclose that both chemically inactivated and recombinant mutant inactive factor IX polypeptides are both to be used in vitro to inhibit thrombus development and because the active site residue mutants of factor IX were known in the prior art, such as that of Ludwig et al. Additionally, Rose et al. explicitly state that "The factor IXa compound may be a recombinant Factor IXa compound in which amino acid**s** (emphasis by examiner) at the active site, especially at the active serine amino acid

site, have been altered to render the recombinant factor IXa functionally inactive, but still capable of competing with intact, native factor IXa for cell surface binding." Given that the active site catalytic triad (Ser365, Asp269, His221) of factor IX was known in the art (see for example the last sentence of the first paragraph in the left column of page 1225 of Ludwig et al.) it is clear that Rose et al. contemplate the use of factor IX in which multiple active site members were simultaneously mutated. Thus the person of ordinary skill in the art would be substituting known equivalents, chemically inactivated and mutant factor IX being equivalent in that both are enzymatically inactive and migrate at the same apparent molecular weight on an SDS-PAGE gel.

Applicant's arguments filed January 26, 2009 have been fully considered but they are not persuasive. Applicant argues that claim 25 as amended does not recite the mutants of factor IX disclosed by Ludwig et al.

This argument is not persuasive because Rose et al. disclose mutant factor IX proteins wherein more than one active site amino acid are mutated, and the three active site amino acids were known in the art at the time the invention was made as disclosed by Ludwig et al. Recited factor IX mutants comprise all possible pairings wherein two of the three positions are mutated, as well as the triple mutation. Given that Rose et al. disclose making active site mutants wherein more than one active site amino acid is mutated and the fact that there are three active site positions, mutating all or any subcombination of the three active site residues is obvious and would result in an enzymatically inactive factor IX polypeptide.

Applicant next argues that the claims of the '403 patent recite inactivated factor IX whereas the instant claims are limited to inactivated factor IXa compounds.

This argument is not persuasive because applicant is arguing limitations not claimed. Specifically, claim 25 as amended 1/26/09 recites both factor IXmi and factor IXami polypeptides. Further, Rose et al. disclose mutant forms of factor IXa.

8. Claim 25 is rejected on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claims 1-19 of U.S. Patent No. 6,316,403 in view of Toledo-Pereyra (of record) in view of Benedict et al. (of record) in view of US Patent 5,839,443 and in view of Ludwig et al. (Blood, 1992, 79:1225-1232).

The claims of the '403 patent recite methods of treating ischemic disorders by administering inactivated factor IX to a patient to inhibit coagulation so as to treat the ischemic disorder in the patient. The claims of the '403 patent differ from the instant claimed invention in that the claims of the '403 patent do not specifically recite reperfusion injury and do not recite that the species of inactivated Factor IX recited in the instant claim.

Toledo-Pereyra discloses that at the time of the instant invention, a skilled artisan would know that "reperfusion injury" is often referred to by the descriptive physiological pathological process of thrombosis (see entire document, particularly the right column of page 1099). Toledo-Pereyra further discloses that fibrinogen activation and clotting (i.e. thrombosis) is a pathophysiological event in reperfusion (see particularly the right column of page 1099 and Table 7 on page 1103).

Benedict et al. disclose that factor IXa inactivated with glu-gly-arg-chloromethyl ketone successfully inhibits thrombus formation in vivo (see entire document, particularly the abstract).

The '443 patent discloses methods of inhibiting thrombus formation in vivo using inactivated factor IXa compounds, such compounds being disclosed as including factor IXa inactivated with glu-gly-arg-chloromethyl ketone as well as recombinant factor IX polypeptides wherein amino acids at the active site, particularly the active site serine, are mutated (see entire document, particularly pages 8 and 9).

Ludwig et al. disclose that the active site of factor IX comprises His221, Asp269 and Ser365, and further discloses multiple mutant factor IX polypeptides that are enzymatically inactive due to substitution mutations at these positions (see entire document, particularly the abstract).

Therefore, it would have been obvious to a person of ordinary skill in the art at the time the invention was made to administer factor IXa compounds, such as

inactivated factor IX and IXa to treat reperfusion injuries. Motivation to do so comes from the teachings of the '403 patent which teaches that "Factor IXa" compounds are to be administered to inhibit thrombosis in a patient, and the teachings of Toledo-Pereyra that thrombosis is an important pathophysiological even that occurs in reperfusion injury. A person of skill in the art would be motivated to use a mutant factor IXa comprising active site mutations since Benedict et al. successfully inhibited thrombus formation in vivo using factor IX chemically inactivated using Glu-Gly-Arg chloromethyl ketone, since the '443 patent discloses that both chemically inactivated and recombinant mutant inactive factor IX polypeptides are both to be used in vitro to inhibit thrombus development and because the active site residue mutants of factor IX were known in the prior art, such as that of Ludwig et al. Additionally, the '443 patent explicitly states that "The factor IXa compound may be a recombinant Factor IXa compound in which amino acid**s** (emphasis by examiner) at the active site, especially at the active serine amino acid site, have been altered to render the recombinant factor IXa functionally inactive, but still capable of competing with intact, native factor IXa for cell surface binding." Given that the active site catalytic triad (Ser365, Asp269, His221) of factor IX was known in the art (see for example the last sentence of the first paragraph in the left column of page 1225 of Ludwig et al.) it is clear that the '443 patent contemplates the use of factor IX in which multiple active site members were simultaneously mutated. Thus the person of ordinary skill in the art would be substituting known equivalents, chemically inactivated and mutant factor IX being equivalent in that both are enzymatically inactive and migrate at the same apparent molecular weight on an SDS-PAGE gel.

Applicant's arguments filed January 26, 2009 have been fully considered but they are not persuasive. The arguments against this double patenting rejection are the same as above because Rose et al. and the '443 patent are the same disclosure, differing from one another only in their pagination and date of availability as prior art. These arguments were not found persuasive for the reasons discussed supra, and thus the rejection is maintained.

9. No claim is allowable.

10. Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

11. Any inquiry concerning this communication or earlier communications from the examiner should be directed to Michael Szperka whose telephone number is (571)272-2934. The examiner can normally be reached on M-F 8:00-4:30.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Eileen O'Hara can be reached on 571-272-0878. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

Michael Szperka, Ph.D.
Primary Examiner
Art Unit 1644

/Michael Szperka, Ph.D./
Primary Examiner, Art Unit 1644